

Double-lumen carotid plaque: A morbid configuration

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During analysis of carotid plaque anatomy for a multicenter carotid imaging trial, we examined plaque specimens from 5 patients with double internal carotid artery lumina. Four of the 5 patients had symptoms referable to the lesion. The second lumen was noted when the plaque specimens were examined *ex vivo* with high-resolution ($200\ \mu\text{m}^3$) magnetic resonance imaging. Plaque structure was correctly identified in only 1 patient preoperatively. However, during retrospective review of the preoperative imaging studies, the second internal carotid artery lumen was identified in 3 patients. (*J Vasc Surg* 2003;37:1314-7.)

Since January 1997 we have conducted a multicenter imaging trial, ACSCEPT (Accuracy of Carotid Stenosis by Correlation with Endarterectomy Plaque Trial). In this trial 320 plaque specimens were excised en bloc and examined *ex vivo* with high-resolution ($200\ \mu\text{m}^3$) magnetic resonance imaging (MRI) to establish degree of stenosis and lumen configuration. During this ongoing analysis of carotid plaque anatomy, we observed 5 cases in which there was a duplicate internal carotid artery lumen. This unusual configuration appeared to have a strong correlation with ipsilateral neurologic events and, to our knowledge, has not been previously reported.

METHODS

Clinical data, ultrasound studies, magnetic resonance angiography (MRA), and conventional angiography were performed preoperatively in all patients. In addition, 1 patient underwent carotid MRA *in vivo* with a custom-made carotid MRA receive-only surface coil.

Plaque specimens were removed en bloc and included the entire atherosclerotic lesion at the carotid bifurcation (common carotid artery, internal carotid artery, and external carotid artery). Specimens were washed to remove adherent blood, immersed in saline solution, and doped with 1:300 parts gadolinium-diethylene-triamine-penta-acetic acid to maximize signal intensity of the medium surrounding the plaque and within the lumen. The prepared specimens were placed in a 2 cm diameter cylinder and imaged with a transmit-receive radiofrequency coil constructed with a small sensitive volume matched closely to the volume of the cylinder containing the plaque. Imaging was performed with a Symphony 1.5 T scanner (Sie-

mens). The principal geometric structural sequence used was a three-dimensional gradient-echo sequence (recovery time, 40 ms; echo time, 12 ms; flip angle, 20 degrees); matrix size, 256×128 , with 64 partitions. The field of view was $50 \times 25 \times 13$ mm in the x, y, and z directions, respectively, with a resulting section thickness of $200\ \mu\text{m}$ and $200\ \mu\text{m} \times 200\ \mu\text{m}$ in plane resolution. The plaque image (high-resolution MRI) was reviewed with NIH Image software (Wayne Rasband, National Institutes of Health, Bethesda, Md).

CASE REPORTS

High resolution MRI of the excised plaques demonstrated a second flow channel in 5 patients.

Case 1. A 63-year-old man reported an episode of tingling extending down the right side of his body and lasting for approximately 5 minutes. This was not associated with any visual disturbance or weakness. He had two episodes with similar symptoms in the previous 4 months. His medications included aspirin. A carotid ultrasound scan was obtained, which showed a moderate degree of stenosis (50%-70%) in the proximal left internal carotid artery. MRA and digital subtraction angiography images verified the lesion and were read as showing plaque irregularity but no ulceration.

Case 2. A 79-year-old man had two episodes of right-sided weakness in the 6 months before admission. He had hypertension and hypercholesterolemia, a 100 pack/y smoking history, and had undergone coronary artery bypass surgery. His medications included warfarin sodium. Doppler ultrasound scans showed critical left carotid stenosis. An MRA showed 85% to 90% stenosis of the left internal carotid artery, and an angiogram showed 90% stenosis with irregular plaque.

Case 3. A 69-year-old man had sudden onset of left arm weakness and dysarthria. The patient reported a right-sided cerebrovascular accident 6 months before admission. A computed tomography scan revealed both a new and an old right-hemispheric lesion. He had a medical history significant for hypertension, peripheral vascular disease, coronary artery disease, and hypercholesterolemia. His medications included aspirin. Carotid ultrasound examination revealed 60% to 79% stenosis of the right internal carotid artery. An MRA and an angiogram were obtained

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Competition of interest: none.

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0741-5214/2003/\$30.00 + 0

doi:10.1016/S0741-5214(02)75345-7

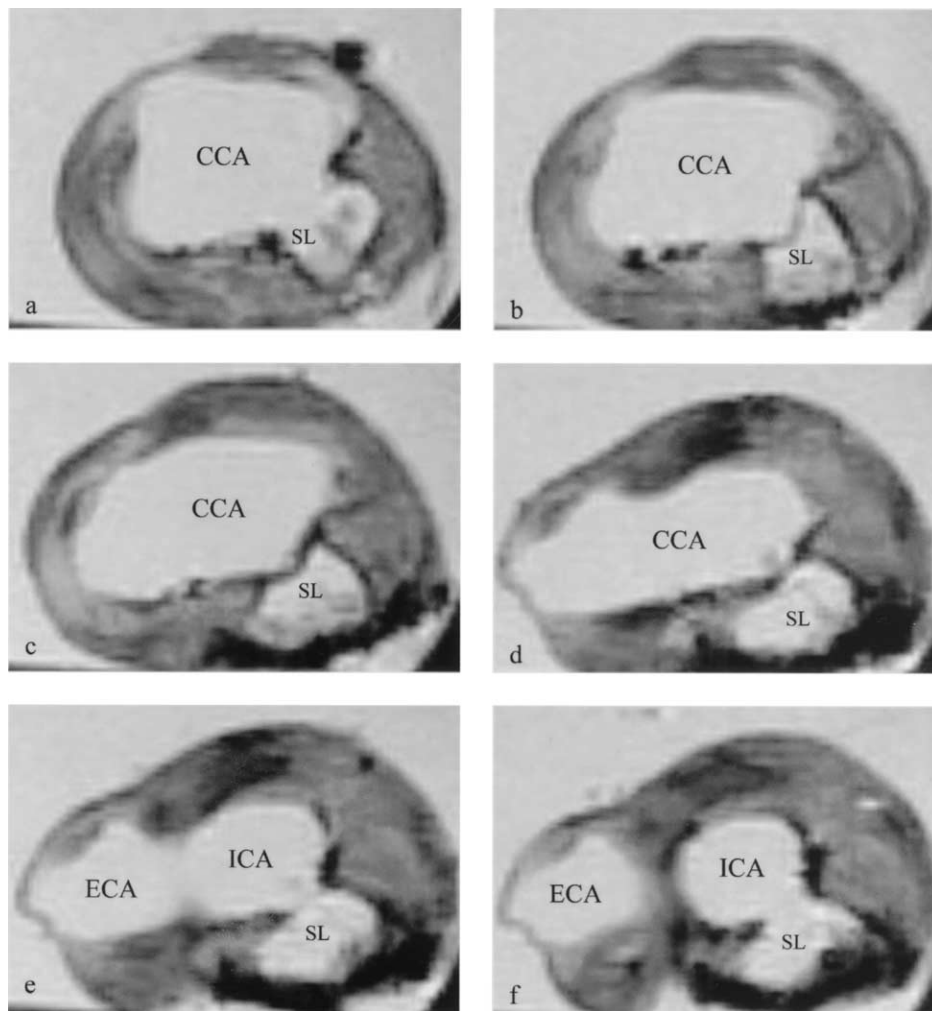


Fig 1. Serial sections through high-resolution MRI images of plaque show a second lumen beginning in the distal common carotid artery (CCA) and reopening at the level of the internal carotid artery (ICA) orifice. SL, Single lumen.

Table I. Characteristics of patients with double-lumen carotid plaque

Patient	Age (y)	Gender	Clinical presentation	Angiography	Magnetic resonance angiography	Specimen magnetic resonance imaging
1	63	Male	TIA	Severe stenosis (70%) of the left ICA, irregular plaque	Left ICA 70%-80% stenosed, irregular appearance in carotid bulb	Double lumen in ICA, with length 8.1 mm and width 2.4 mm
2	79	Male	TIA	Left ICA 90% stenosed, irregular plaque	Left ICA 85%-90% stenosed irregular plaque	Double lumen in the ICA, with length 11.2 mm and width 3.0 mm
3	70	Male	CVA	right ICA 80%-85% stenosed, ulcerated plaque	Proximal right ICA stenosed 80%	Double lumen in ICA, with length 10.96 mm and width 3.3 mm
4	63	Male	Asx	Right ICA 90% stenosed, moderate sized proximal ulceration	Bilateral 80% stenosis of proximal ICA	Double lumen in CCA and ICA, with length 11.9 mm and width 2.4 mm
5	75	Male	TIA	Left ICA 60% stenosed, complex ulcer	Left ICA, "complex plaque" with 80% stenosis and second lumen seen	Double lumen in CCA and ICA, with length 11.4 mm and width 3.1 mm

TIA, Transient ischemic attack; ICA, internal carotid artery; CVA, cerebrovascular accident; Asx, asymptomatic; CCA, common carotid artery.



Fig 2. Magnetic resonance angiogram of case 5 clearly shows two flow channels in the internal carotid artery. A receive-only neck coil was used in this study.

and were interpreted as showing approximately 80% stenosis with ulcerated plaque.

Case 4. A 63-year-old man had prominent carotid bruits at routine examination, but he denied any neurologic symptoms. His medications included aspirin. A carotid duplex scan showed critical stenosis of the right proximal internal carotid artery. Both an MRA and an angiogram demonstrated 80% to 90% stenosis with moderate ulceration of the plaque.

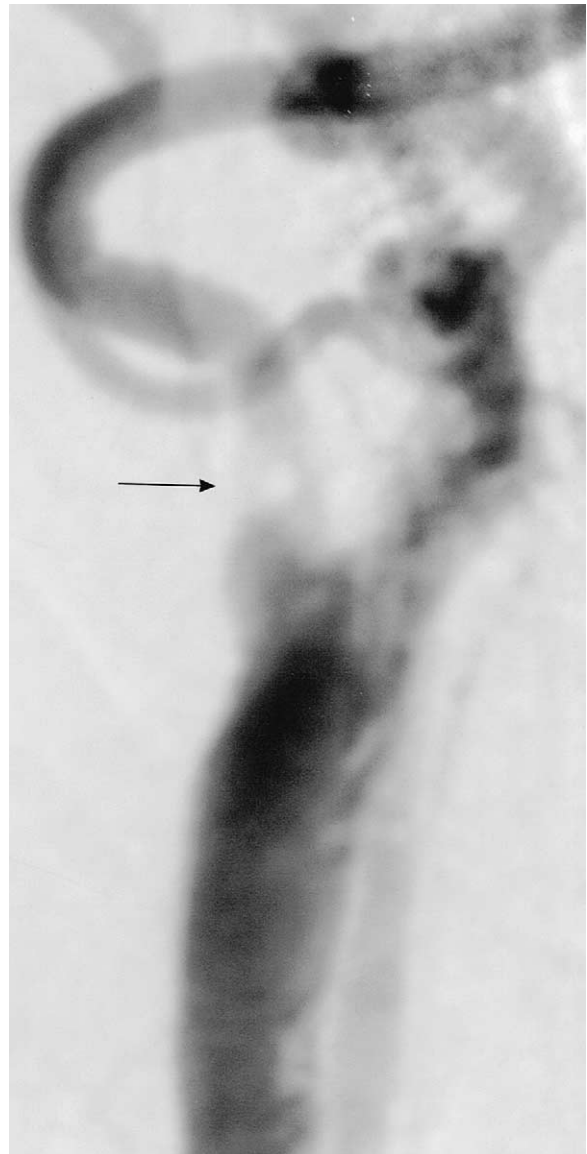


Fig 3. Angiogram of case 3 was originally interpreted as demonstrating a "complex" luminal configuration. In retrospect, a second channel is seen.

Case 5. A 75-year-old man had right arm paralysis. He had undergone left carotid endarterectomy 6 years before admission. His medications included aspirin and warfarin sodium. An ultrasound scan showed recurrent stenosis of approximately 80% in the left internal carotid artery. An angiogram confirmed the stenosis and demonstrated plaque irregularity with a question of ulceration. The MRA, obtained with a surface coil, showed a complex plaque with a second lumen.

RESULTS

The second lumen extended from the common carotid artery to the internal carotid artery in 2 patients and was contained in the internal carotid artery in 3 patients. Aver-

age length of the second lumen was 10.7 ± 1.5 mm, and average width was 2.83 ± 0.4 mm (Table). The two flow channels often were separated by a thin (<1 mm) membrane, presumed to be the plaque fibrous cap (Fig 1, A-E).

Preoperatively, even when a double lumen was not appreciated, all plaques were seen as either irregular or ulcerated at both MRA and angiography. The only case in which the plaque anatomy was accurately identified was in the only patient imaged with a carotid surface coil (Fig 2). However, when we retrospectively reviewed the preoperative MRA and angiographic studies for the 4 other patients, we found that a second lumen was imaged but not recognized by the clinicians in 2 of 4 cases (Fig 3).

CONCLUSIONS

We describe an extreme abnormality of plaque anatomy, a channel dissecting through the atherosclerotic plaque, effectively forming a second lumen. Reliably identifying this second lumen requires high-resolution imaging. This entity was originally observed with *ex vivo* high-resolution ($200 \mu\text{m}^3$) MRI. In the ACSCEPT database of 320 carotid plaques, we found 5 with two lumina, for an incidence of 1.6%. Our only *in vivo* (preoperative) detection was achieved with a surface coil with a resolution of 1 mm. However, in review, 3 of the 5 plaque “dissections” were observable on angiograms or routine MRAs but were not recognized by the treating clinicians.

Reliable detection of the double-lumen structure *in vivo* requires high-resolution imaging techniques. Flow through the second lumen was slow and disordered in the 1 case we observed *in vivo*, and no hint of its presence was seen on ultrasound scans. The proximity of the two lumina makes them difficult to visualize individually on angiograms. Higher resolution imaging with either surface coil MRA or CT angiography may enable visualization of these abnormalities in the future. Reviewing MRI or CT images (acquired in cross-section) also will facilitate observation of the second flow channel.

Double-lumen carotid plaque lesions should not be confused with spontaneous carotid dissections. These occur typically in young patients without atherosclerosis. They are often associated with local pain, and may appear as classic beaklike narrowing on imaging studies. Less com-

monly the dissection can create a second lumen, but this generally occurs without significant narrowing.¹ The lesions in our series were associated with significant stenosis and were in older patients who did not volunteer a history of neck pain.

The rate of neurologic symptoms in these cases (80%) suggests that duplicate carotid lumina are not just a novelty of improved carotid imaging. These channels represent a dissection through the plaque necrotic core, presumably originating in an ulceration. The re-entry into the true lumen may enable embolization of plaque core contents or thrombotic material formed on the highly irregular surface of the second lumen. This carotid plaque morphologic abnormality appears to identify a lesion with high risk for associated emboli and stroke.

There is strong evidence that plaque anatomy is an important factor in determining risk for stroke associated with symptomatic carotid atherosclerosis.²⁻⁴ The evidence supporting plaque structure as an indicator of risk in patients without symptoms is less robust. Development of high-resolution technologies for *in vivo* study of plaque anatomy will allow prospective trials of asymptomatic plaques to determine whether certain plaque features indicate high-risk lesions. Until such trials are completed, the suspicion of increased risk for embolization in patients without symptoms with various abnormalities of plaque structure, including duplicate lumen, remains conjecture.

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Submitted Jun 27, 2002; accepted Oct 31, 2002.